

Remarks

Applicants have amended Claims 5, 10 and 20 to limit the claimed assay to a method which uses a membrane containing the IKr channel derived from a cell line transfected with the human ERG gene. In view of this limitation, claims 6, 8, 11, 13, 21, 23 and 25 do not further limit the claims on which they depend, and, accordingly, they have been canceled. Claims 7, 9, 12, 14, 19, 22, 24, and 26, which previously depended on the now-canceled claims, have been amended to depend on currently pending claims.

The Examiner rejected Claims 5-27 under 35 USC 103(a) as unpatentable over a) Baldwin, et al, in combination with each of b) Chadwick, et al., Fiset, et al., Geonzon, et al., or Duff, et al., and with c) Dean, et al. The Examiner sets forth reasons for the rejection in the office action, and also refers to reasons presented in the October 20, 2003 final rejection and January 26, 2004 advisory action.

The Examiner indicated that Baldwin, et al. describes the non-radiolabeled, sulfonamide compound of formula I of Claim 1, that each of Chadwick, et al., Fiset, et al., Geonzon, et al., and Duff, et al. describe assays that assess the membrane K⁺ channel blocking activity of radiolabeled [³H] dofetilide, and that Dean, et al. describe use of ³⁵S-containing sulfonamide groups for sulfonamide-containing ligands used in receptor binding radioassays. The Examiner stated in the advisory action that the specification indicates that Chadwick, et al., Fiset, et al., Geonzon, et al., and Duff, et al. describe assays involving "screening for the same type of IKr activity which is involved in the instantly claimed methods".

The specification states that these references describe IKr assays conducted using a [³H]-radioligand capable of binding to ERG in guinea pig monocytes or patch clamping on intact cells. However, the presently claimed invention is distinct from the previous methods.

The presently claimed binding assay uses membranes from cells expressing human ERG, rather than ERG in intact animal cells, as the source of the ion channel. Use of human ERG in the present assay provides binding information that is relevant to understanding binding to human ERG. Assays using guinea pig monocytes or patch clamping on intact cells do not provide binding information representative of binding to human ERG. Furthermore, Chadwick, et al., Fiset, et al., Geonzon, et al., and Duff, et al. do not describe or suggest a binding assay using membranes from cells expressing human ERG.

In view of the amendment of Claims 5, 10 and 20 to limit the claimed assay to a method which uses a membrane containing the IKr channel derived from a cell line transfected with the human ERG gene, cancellation of claims 6, 8, 11, 13, 21, 23 and 25, and amendment of Claims 7,

JUL-02-2004 13:18

732 594 1590

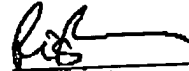
P.08

USSN 09/904,045
Examiner M. Ceperley
page 7

9, 12, 14, 19, 22, 24, and 26, applicants believe the application is in condition for allowance.

Respectfully submitted,

Date: July 2, 2004



Richard S. Parr
Attorney for Applicants
MERCK & CO., Inc.
P.O. Box 2000
Rahway, NJ 07065-0907
Tel.: (732) 594-4958